MECHANICS OF COCHLEAR OUTER HAIR CELLS AND STEREOCILIA

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There are around 10,000 outer hair cells in the mammalian cochlea. These cells are roughly cylindrical in shape, 10 μm in diameter and 40-70μm in length [1]. At the apical pole of the cell are 100 or so stereocilia organized in three rows. Each stereocilia is several μm in length, 500 nm in diameter. The rows of stereocilia are connected at the tips by a link comprised of a double helix protein 8-12 nm in diameter and around 100 nm long [2]. These tip links are thought be nearly rigid. Through the shearing action of the hair bundle the tip links are affect the channel opening that allows ions to pass into the the OHC cell body[1]. As a whole (OHC soma and hair bundles), the OHC are hypothesized to provide cycle-by-cycle amplification of low level acoustic signals in the cochlea by converting resting potentials into mechanical forces at frequencies up to 100,000 kHz. Just how this is accomplished in a biological system has been an open quesiton in biology for many years. A protein in the outer hair cell wall, prestin, is necessary for somatic transduction of fluctuating transmembrane voltage into mechanical motility. Such transduction is unique to mammals and has been shown to exist up to 100 kHz *in vivo* [3]. Hair bundle motility is seen in lower vertebrates as well as mammals. In mammals, this motility, putatively based on channel closure and reopening in response to a calcium binding event, has been seen to occur at time constants near 100μs, nearly fast enough to contribute to cycle-by-cycle transduction over the hearing range in mammals [4]. In this talk we present models for OHC electromotility motility based on the probability of open and closed stereocilia channels coupled to nonlinear models of the OHC soma. Further, we examine the effectiveness of each type of motility as a mediator of amplification, by studying the interaction of the OHC with the surrounding fluid and mechanical environment.

References

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